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 NEWS  8  DEC 15  MEDLINE update schedule for December 2004
 NEWS  9  DEC 17  ELCOM reloaded; updating to resume; current-awareness
                  alerts (SDIs) affected
 NEWS  10 DEC 17  COMPUAB reloaded; updating to resume; current-awareness
                  alerts (SDIs) affected
 NEWS  11 DEC 17  SOLIDSTATE reloaded; updating to resume; current-awareness
                  alerts (SDIs) affected
 NEWS  12 DEC 17  CERAB reloaded; updating to resume; current-awareness
                  alerts (SDIs) affected
 NEWS  13 DEC 17  THREE NEW FIELDS ADDED TO IFIPAT/IFIUDB/IFICDB
 NEWS  14 DEC 30  EPFULL: New patent full text database to be available on STN
 NEWS  15 DEC 30  CAPLUS - PATENT COVERAGE EXPANDED
 NEWS  16 JAN 03  No connect-hour charges in EPFULL during January and
                  February 2005
 NEWS  17 JAN 26  CA/CAPLUS - Expanded patent coverage to include the Russian
                  Agency for Patents and Trademarks (ROSPATENT)
 NEWS  18 FEB 10  STN Patent Forums to be held in March 2005
 NEWS  19 FEB 16  STN User Update to be held in conjunction with the 229th ACS
                  National Meeting on March 13, 2005

 NEWS EXPRESS  JANUARY 10 CURRENT WINDOWS VERSION IS V7.01a, CURRENT
               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005

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FILE COVERS 1907 - 19 Feb 2005  VOL 142 ISS 9
FILE LAST UPDATED: 18 Feb 2005  (20050218/ED)

  This file contains CAS Registry Numbers for easy and accurate
  substance identification.


=> reovirus
          1881 REOVIRUS
           313 REOVIRUSES
L1        1946 REOVIRUS
                 (REOVIRUS OR REOVIRUSES).

=> reassorted
L2          45 REASSORTED

=> L1 and L2
L3           1 L1 AND L2

=> reassortant and l1
           461 REASSORTANT
           324 REASSORTANTS
           614 REASSORTANT
                 (REASSORTANT OR REASSORTANTS)
L4          67 REASSORTANT AND L1

=> propagation and l4
        115130 PROPAGATION
           479 PROPAGATIONS
        115378 PROPAGATION
                 (PROPAGATION OR PROPAGATIONS)
L5           1 PROPAGATION AND L4

=> production and L4
        546397 PRODUCTION
          2701 PRODUCTIONS
        548367 PRODUCTION
                 (PRODUCTION OR PRODUCTIONS)
        856911 PRODN
           528 PRODNS
```

857091 PRODN
(PRODN OR PRODNS)
1177333 PRODUCTION
(PRODUCTION OR PRODN)
L6 6 PRODUCTION AND L4

=> D L6 IBIB ABS 1-6

L6 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:483999 CAPLUS
DOCUMENT NUMBER: 141:64475
TITLE: Inhibition of **reovirus** by mycophenolic acid is associated with the M1 genome segment
AUTHOR(S): Hermann, Laura L.; Coombs, Kevin M.
CORPORATE SOURCE: Department of Medical Microbiology and Infectious Diseases and Department of Physiology, University of Manitoba, Winnipeg, MB, R3E 0W3, Can.
SOURCE: Journal of Virology (2004), 78(12), 6171-6179
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Mycophenolic acid (MPA), an inhibitor of IMP dehydrogenase, inhibits **reovirus** replication and viral RNA and protein **prodn**. In mouse L929 cells, antiviral effects were greatest at 30 μg of MPA/mL. At this dosage, MPA inhibited replication of **reovirus** strain T3D more than 1,000-fold and inhibited replication of **reovirus** strain T1L nearly 100-fold, compared to non-drug-treated controls. Genetic **reassortant** anal. indicated the primary determinant of strain-specific differences in sensitivity to MPA mapped to the viral M1 genome segment, which encodes the minor core protein μ2. MPA also inhibited replication of both strains of **reovirus** in a variety of other cell lines, including Vero monkey kidney and U373 human astrocytoma cells. Addition of exogenous guanosine to MPA-treated **reovirus**-infected cells restored viral replicative capacity to nearly normal levels. These results suggest the μ2 protein is involved in the uptake and processing of GTP in viral transcription in infected cells and strengthens the evidence that the μ2 protein can function as an NTPase and is likely a transcriptase cofactor.
REFERENCE COUNT: 72 THERE ARE 72 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2001:361260 CAPLUS
DOCUMENT NUMBER: 135:136307
TITLE: Avian **reovirus** major μ-class outer capsid protein influences efficiency of productive macrophage infection in a virus strain-specific manner
AUTHOR(S): O'Hara, David; Patrick, Megan; Cepica, Denisa; Coombs, Kevin M.; Duncan, Roy
CORPORATE SOURCE: Department of Microbiology and Immunology, Dalhousie University, Halifax, NS, B3H 4H7, Can.
SOURCE: Journal of Virology (2001), 75(11), 5027-5035
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We determined that the highly pathogenic avian **reovirus** strain 176 (ARV-176) possesses an enhanced ability to establish productive infections in HD-11 avian macrophages compared to avian fibroblasts. Conversely, the weakly pathogenic strain ARV-138 shows no such macrophagotropic tendency. The macrophage infection capability of the two viruses did not reflect differences in the ability to either induce or inhibit nitric oxide **prodn**. Moderate increases in the ARV-138 multiplicity of

infection resulted in a concomitant increase in macrophage infection, and under such conditions the kinetics and extent of the ARV-138 replication cycle were equivalent to those of the highly infectious ARV-176 strain. These results indicated that both viruses are apparently equally capable of replicating in an infected macrophage, but they differ in the ability to establish productive infections in these cells. Using a genetic **reassortant** approach, we determined that the macrophagotropic property of ARV-176 reflects a post-receptor-binding step in the virus replication cycle and that the ARV-176 M2 genome segment is required for efficient infection of HD-11 cells. The M2 genome segment encodes the major μ-class outer capsid protein (μB) of the virus, which is involved in virus entry and transcriptase activation, suggesting that a host-specific influence on ARV entry and/or uncoating may affect the likelihood of the virus establishing a productive infection in a macrophage cell.

REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1997:121692 CAPLUS
DOCUMENT NUMBER: 126:196712
TITLE: Characterization of an ATPase activity in **reovirus** cores and its genetic association with core-shell protein λ1
AUTHOR(S): Noble, Simon; Nibert, Max L.
CORPORATE SOURCE: Inst. Mol. Virol. Dep. Biochem., Univ. Wisconsin-Madison, Madison, WI, USA
SOURCE: Journal of Virology (1997), 71(3), 2182-2191
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A previously identified nucleoside triphosphatase activity in mammalian **reovirus** cores was further characterized by comparing two **reovirus** strains whose cores differ in their efficiencies of ATP hydrolysis. In assays using a panel of **reassortant** viruses derived from these strains, the difference in ATPase activity at standard conditions was genetically associated with viral genome segment L3, encoding protein λ1, a major constituent of the core shell that possesses sequence motifs characteristic of other ATPases. The ATPase activity of cores was affected by several other reaction components, including temperature, pH, nature and concentration of monovalent and divalent cations, and nature and concentration of anions. A strain difference in the response of core ATPase activity to monovalent acetate salts was also mapped to L3/λ1 by using **reassortant** viruses. Expts. with different nucleoside triphosphates demonstrated that ATP is the preferred ribonucleotide substrate for cores of both strains. Other expts. suggested that the ATPase is latent in **reovirus** virions and infectious subviral particles but undergoes activation during **prodn.** of cores in close association with the protease-mediated degradation of outer-capsid protein μ1 and its cleavage products, suggesting that μ1 may play a role in regulating the ATPase.

REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1996:440965 CAPLUS
DOCUMENT NUMBER: 125:84835
TITLE: Method for producing biologicals in protein-free culture
INVENTOR(S): Kistner, Otfried; Barrett, Noel; Mundt, Wolfgang; Dorner, Friedrich
PATENT ASSIGNEE(S): Immuno Aktiengesellschaft, Austria
SOURCE: PCT Int. Appl., 97 pp.

CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9615231	A2	19960523	WO 1995-EP4439	19951110
WO 9615231	A3	19960801		
W: CA, FI, JP, NO, US				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 5753489	A	19980519	US 1995-487046	19950607
US 5756341	A	19980526	US 1995-483522	19950607
EP 791055	A1	19970827	EP 1995-937888	19951110
R: AT, BE, CH, DE, DK, ES, FR, GB, IE, IT, LI, NL, SE				
JP 10503093	T2	19980324	JP 1995-515726	19951110
FI 9701998	A	19970509	FI 1997-1998	19970509

PRIORITY APPLN. INFO.:

US 1994-338761	A	19941110
US 1995-483522	A	19950607
US 1995-487046	A	19950607
US 1995-487222	A	19950607
WO 1995-EP4439	W	19951110

AB The present invention includes an approach for producing viruses, such as influenza, and vaccines derived therefrom as well as recombinant proteins derived from viral vectors, by utilizing vertebrate cells cultured under protein-free conditions. These cells, which include a cellular biomass, show improved capabilities for propagating viruses and eliminate the need for costly and time-consuming viral passaging and purification The invention also includes further approaches for enhancing the propagation of viruses by employing activating substances, modifying the activation site of viruses, and using augmentation loops. Improved approaches for producing viral **reassortants** also are provided.

L6 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1995:374535 CAPLUS
DOCUMENT NUMBER: 122:157573
TITLE: **Reovirus** mutant tsA279 has temperature-sensitive lesions in the M2 and L2 genes and association of M2 gene with decreased viral protein **production** and blockage in transmembrane transport
AUTHOR(S): Hazelton, Paul R.; Coombs, Kevin M.
CORPORATE SOURCE: Dep. Med. Microbiol. Infectious Diseases, Univ. Manitoba, Winnipeg, MB, R3E 0W3, Can.
SOURCE: Virology (1995), 207(1), 46-58
CODEN: VIRLAX; ISSN: 0042-6822
PUBLISHER: Academic
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Temperature-sensitive mutants provide an ideal means for dissecting viral assembly pathways. The morphol. variants produced by and biol. characteristics of tsA279, a previously uncharacterized mutant from the Fields' panel of temperature-sensitive mutants of **reovirus**, were determined under restrictive growth conditions. The mutant showed a distinctive pattern of increased temperature sensitivity as the temperature was raised from 39° to 40°. Wild-type **reovirus** type 1 Lang and the mutant were crossed to generate **reassortants**. Efficiency of plating analyses of the **reassortants** showed that tsA279 has temperature-sensitive lesions in two genes, a mildly temperature-sensitive one in L2, which encodes core spike protein λ2, and a stronger, dominant lesion in M2, which encodes major outer capsid protein μ1. Electron microscopic examination of thin-sectioned tsA279-infected cells showed three

ways in which the mutant phenotypes were expressed. The mutant appeared to be blocked in transmembrane transport of virions, a phenotype that mapped to the M2 gene; the mutant produced significantly reduced amts. of identifiable particles; and those particles that were produced appeared to be morphol. variants. Immunofluorescent microscopy and immunopptns. of tsA279- and various T1L x tsA279 **reassortant**-infected cells suggested that the reduction in observed progeny was caused by a decreased **prodn**. of viral proteins at the nonpermissive temperature This phenotype also mapped to the mutant M2 gene.

L6 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1993:76880 CAPLUS
DOCUMENT NUMBER: 118:76880
TITLE: Strain-specific selection of genome segments in avian **reovirus** coinfections
AUTHOR(S): Ni, Yawei; Kemp, Maurice C.
CORPORATE SOURCE: Coll. Vet. Med., Texas A and M Univ., College Station, TX, 77843, USA
SOURCE: Journal of General Virology (1992), 73(12), 3107-13
CODEN: JGVIAY; ISSN: 0022-1317
DOCUMENT TYPE: Journal
LANGUAGE: English
AB To determine whether selection of genome segments in coinfections is strain-specific, chicken embryo fibroblasts were coinfected with avian **reovirus** strain 883 and one of three other avian **reovirus** strains (176, S1133 and 81-5). Viral progeny from each coinfection (883 + 176, 883 + S1133 or 883 + 81-5) was serially passaged at a low m.o.i. The electropherotypes of the coinfection progeny and those of the plaque-derived clones obtained from passages 1 and 20 were analyzed. Two 883 segments (M2 and S2) were found to be selected in the 883 + 176 coinfection, three 883 segments (M2, M3, and S2) in the 883 + S1133 coinfection, and only one 883 segment (M3) in the 883 + 81-5 coinfection, i.e. different 883 genome segments were selected in the 3 coinfections. It was, therefore, concluded that selection of genome segments in a coinfection of a given cell line is virus strain-specific. The selection of genome segments in coinfections was shown to be due to enhanced infectivity of the **reassortants** that were formed in the coinfections. In addition, defective interfering particles that lack the S1 segment were identified in the 883 + 81-5 coinfection progeny following serial passage. Selection of genome segment(s) in coinfections as described herein may have potential importance on the effect and **prodn**. of divalent or multivalent vaccines.

=> D L5 IBIB ABs

L5 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1996:440965 CAPLUS
DOCUMENT NUMBER: 125:84835
TITLE: Method for producing biologicals in protein-free culture
INVENTOR(S): Kistner, Otfried; Barrett, Noel; Mundt, Wolfgang; Dorner, Friedrich
PATENT ASSIGNEE(S): Immuno Aktiengesellschaft, Austria
SOURCE: PCT Int. Appl., 97 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

WO 9615231	A2	19960523	WO 1995-EP4439		19951110
WO 9615231	A3	19960801			
W: CA, FI, JP, NO, US					
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE					
US 5753489	A	19980519	US 1995-487046		19950607
US 5756341	A	19980526	US 1995-483522		19950607
EP 791055	A1	19970827	EP 1995-937888		19951110
R: AT, BE, CH, DE, DK, ES, FR, GB, IE, IT, LI, NL, SE					
JP 10503093	T2	19980324	JP 1995-515726		19951110
FI 9701998	A	19970509	FI 1997-1998		19970509
PRIORITY APPLN. INFO.:			US 1994-338761	A	19941110
			US 1995-483522	A	19950607
			US 1995-487046	A	19950607
			US 1995-487222	A	19950607
			WO 1995-EP4439	W	19951110

AB The present invention includes an approach for producing viruses, such as influenza, and vaccines derived therefrom as well as recombinant proteins derived from viral vectors, by utilizing vertebrate cells cultured under protein-free conditions. These cells, which include a cellular biomass, show improved capabilities for propagating viruses and eliminate the need for costly and time-consuming viral passaging and purification The invention also includes further approaches for enhancing the **propagation** of viruses by employing activating substances, modifying the activation site of viruses, and using augmentation loops. Improved approaches for producing viral **reassortants** also are provided.

=> D L3 IBIB ABS

L3 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER:	1995:851366 CAPLUS
DOCUMENT NUMBER:	123:248214
TITLE:	High resolution genome typing and genomic reassortment events of rice dwarf Phytoreovirus
AUTHOR(S):	Uyeda, Ichiro; Ando, Yuko; Murao, Kazunori; Kimura, Ikuo
CORPORATE SOURCE:	Fac. Agriculture, Hokkaido Univ., Sapporo, 060, Japan
SOURCE:	Virology (1995), 212(2), 724-7 CODEN: VIRLAX; ISSN: 0042-6822
PUBLISHER:	Academic
DOCUMENT TYPE:	Journal
LANGUAGE:	English

AB Genomic reassortment of rice dwarf Phytoreovirus (RDV) was exptly. demonstrated for the first time in plant **reoviruses**. Combinations of two genomic variants, most of the genomic segments of which could be distinguished by a high resolution polyacrylamide gel electrophoresis, were used to produce genomic reassortants. After artificial mixed injection of two of three isolates (RDV-S, RDV-Al, and RDV-AN) into the insect vector Nephotettix cincticeps, rice seedlings were sequentially inoculated and the genomic origin of the viruses present in the infected plants were examined by electrophoresis. The progeny virus population contained either one or both of the resp. genomic segments from the parents. Genomic segments **reassorted** randomly except for genome segment 1 (S1) and S9. S9 of RDV-s was mostly excluded in the reassortants in both the insects and the infected plants when it was mixed with RDV-Al or RDV-AN. On the other hand, S9 **reassorted** randomly in most of the virus populations in infected plants when RDV-Al and RDV-AN were co-injected into insects. When RDV-S and RDV-Al were mixed, S1 from RDV-S was present more frequently in the infected plants although both parental S1's were present in equimolar amts. in insects.

=> reovirus (l) reassorted
1881 REOVIRUS

```
           313 REOVIRUSES
          1946 REOVIRUS
                 (REOVIRUS OR REOVIRUSES)
            45 REASSORTED
L7           1 REOVIRUS (L) REASSORTED

=> reovirus (l) reassortant
          1881 REOVIRUS
           313 REOVIRUSES
          1946 REOVIRUS
                 (REOVIRUS OR REOVIRUSES)
           461 REASSORTANT
           324 REASSORTANTS
           614 REASSORTANT
                 (REASSORTANT OR REASSORTANTS)
L8          65 REOVIRUS (L) REASSORTANT

=> reovirus (p) reassortant
          1881 REOVIRUS
           313 REOVIRUSES
          1946 REOVIRUS
                 (REOVIRUS OR REOVIRUSES)
           461 REASSORTANT
           324 REASSORTANTS
           614 REASSORTANT
                 (REASSORTANT OR REASSORTANTS)
L9          65 REOVIRUS (P) REASSORTANT

=> reovirus (s0 reassortant
MISSING OPERATOR 'REOVIRUS (S0'
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> reovirus (s) reassortant
          1881 REOVIRUS
           313 REOVIRUSES
          1946 REOVIRUS
                 (REOVIRUS OR REOVIRUSES)
           461 REASSORTANT
           324 REASSORTANTS
           614 REASSORTANT
                 (REASSORTANT OR REASSORTANTS)
L10         27 REOVIRUS (S) REASSORTANT

=> D l10 IBIB ABS 1-27

L10  ANSWER 1 OF 27  CAPLUS  COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER:        2002:972033  CAPLUS
DOCUMENT NUMBER:         138:234613
TITLE:                   The M2 gene segment is involved in the capacity of
                         reovirus type 3 Abney to induce the oily fur syndrome
                         in neonatal mice, a S1 gene segment-associated
                         phenotype
AUTHOR(S):               Derrien, Muriel; Hooper, Jay W.; Fields, Bernard N.
CORPORATE SOURCE:        Dep. Microbiol. Mol. Genet., Harvard Med. Sch.,
                         Boston, MA, 02115, USA
SOURCE:                  Virology (2002), Volume Date 2003, 305(1), 25-30
                         CODEN: VIRLAX; ISSN: 0042-6822
PUBLISHER:               Elsevier Science
DOCUMENT TYPE:           Journal
LANGUAGE:                English
AB   Oral inoculation of reovirus type 3 Abney (T3A) into neonatal mice induces
     hepatitis and the biliary atresia-associated oily fur syndrome (OFS), a
     phenotype previously linked to the S1 gene.  We found that following oral
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inoculation, none of three T3A mutants, JH2, JH3, and JH4, containing different single amino acid substitutions in the M2 gene, induced the OFS or extensive liver necrosis. Similarly, **reassortant** viruses containing both a JH4-S1 and a JH4-M2 gene segment did not induce the OFS, whereas another **reassortant** containing a JH4-S1 gene and a M2 gene from **reovirus** type 3 Dearing fully recovered this capacity. Together, these results constitute the first evidence for the involvement of the M2 gene in the S1 gene-associated capacity of T3A to induce hepatobiliary disease in neonatal mice.

REFERENCE COUNT: 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 2 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2002:426715 CAPLUS
TITLE: Oncolytic virus
INVENTOR(S): Brown, Earl Garnet; Mbisa, Jean Lutamyo; Bell, John Cameron; Stodjl, David Francis
PATENT ASSIGNEE(S): University of Ottawa, Can.
SOURCE: PCT Int. Appl.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002043647	A2	20020606	WO 2001-CA1703	20011130
WO 2002043647	A3	20030103		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2430495	AA	20020606	CA 2001-2430495	20011130
AU 2002020416	A5	20020611	AU 2002-20416	20011130
EP 1339736	A2	20030903	EP 2001-998300	20011130
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2004519431	T2	20040702	JP 2002-545626	20011130
WO 2002050304	A2	20020627	WO 2001-US45108	20011203
WO 2002050304	A3	20021128		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002043257	A5	20020701	AU 2002-43257	20011203
US 2004115170	A1	20040617	US 2004-433064	20040108

PRIORITY APPLN. INFO.:		
US 2000-250131P	P	20001201
US 2001-327016P	P	20011005
WO 2001-CA1703	W	20011130
WO 2001-US45108	W	20011203

AB Methods of reducing the viability of a tumor cell, infecting a neoplasm in a mammal, utilizing certain non-naturally occuring viruses are disclosed. Viral **reassortants**, for example **reovirus**

reassortants, and techniques for identifying PKR-sensitive viruses are also disclosed.

L10 ANSWER 3 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2002:333922 CAPLUS
DOCUMENT NUMBER: 137:75660
TITLE: Sites and determinants of early cleavages in the proteolytic processing pathway of reovirus surface protein σ3
AUTHOR(S): Jane-Valbuena, Judit; Breun, Laura A.; Schiff, Leslie A.; Nibert, Max L.
CORPORATE SOURCE: Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, 02115, USA
SOURCE: Journal of Virology (2002), 76(10), 5184-5197
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Entry of mammalian reovirus virions into target cells requires proteolytic processing of surface protein σ3. In the virion, σ3 mostly covers the membrane-penetration protein μ1, appearing to keep it in an inactive form and to prevent it from interacting with the cellular membrane until the proper time in infection. The mol. mechanism by which σ3 maintains μ1 in this inactive state and the structural changes that accompany σ3 processing and μ1 activation, however, are not well understood. In this study we characterized the early steps in σ3 processing and determined their effects on μ1 function and particle infectivity. We identified 2 regions of high protease sensitivity, "hypersensitive" regions located at residues 208-214 and 238-244, within which all proteases tested selectively cleaved σ3 as an early step in processing. Further processing of σ3 was required for infection, consistent with the fact that the fragments resulting from these early cleavages remained bound to the particles. **Reovirus** type 1 Lang (T1L), type 3 Dearing (T3D), and T1L + T3D **reassortant** virions differed in the sites of early σ3 cleavage, with T1L σ3 being cleaved mainly at residues 238-244 and T3D σ3 being cleaved mainly at residues 208-214. These virions also differed in the rates at which the early cleavages occurred, with cleavage of T1L σ3 occurring faster than cleavage of T3D σ3. Analyses using chimeric and site-directed mutants of recombinant σ3 identified carboxy-proximal residues 344, 347, and 353 as the primary determinants of these strain differences. The spatial relationships between these more carboxy-proximal residues and the hypersensitive regions were discerned from the σ3 crystal structure. The results indicate that proteolytic processing of σ3 during reovirus disassembly is a multistep pathway with a number of mol. determinants.
REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 4 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2001:832873 CAPLUS
DOCUMENT NUMBER: 136:117240
TITLE: Reovirus infection activates JNK and the JNK-dependent transcription factor c-Jun
AUTHOR(S): Clarke, Penny; Meintzer, Suzanne M.; Widmann, Christian; Johnson, Gary L.; Tyler, Kenneth L.
CORPORATE SOURCE: Department of Neurology, University of Colorado Health Science Center, Denver, CO, 80262, USA
SOURCE: Journal of Virology (2001), 75(23), 11275-11283
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Viral infection often perturbs host cell signaling pathways including

those involving mitogen-activated protein kinases (MAPKs). We now show that reovirus infection results in the selective activation of c-Jun N-terminal kinase (JNK). Reovirus-induced JNK activation is associated with an increase in the phosphorylation of the JNK-dependent transcription factor c-Jun. Reovirus serotype 3 prototype strains Abney (T3A) and Dearing (T3D) induce significantly more JNK activation and c-Jun phosphorylation than does the serotype 1 prototypic strain Lang (T1L). T3D and T3A also induce more apoptosis in infected cells than T1L, and there was a significant correlation between the ability of these viruses to phosphorylate c-Jun and induce apoptosis. However, reovirus-induced apoptosis, but not reovirus-induced c-Jun phosphorylation, is inhibited by blocking TRAIL/receptor binding, suggesting that apoptosis and c-Jun phosphorylation involve parallel rather than identical pathways. Strain-specific differences in JNK activation are determined by the reovirus S1 and M2 gene segments, which encode viral outer capsid proteins (σ1 and μ1c) involved in receptor binding and host cell membrane penetration. These same gene segments also determine differences in the capacity of **reovirus** strains to induce apoptosis, and again a significant correlation between the capacity of T1L + T3D **reassortant reoviruses** to both activate JNK and phosphorylate c-Jun and to induce apoptosis was shown. The extracellular signal-related kinase (ERK) is also activated in a strain-specific manner following reovirus infection. Unlike JNK activation, ERK activation could not be mapped to specific reovirus gene segments, suggesting that ERK activation and JNK activation are triggered by different events during virus-host cell interaction.

REFERENCE COUNT: 63 THERE ARE 63 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 5 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1999:181455 CAPLUS
DOCUMENT NUMBER: 131:1325
TITLE: The reovirus mutant tsA279 L2 gene is associated with generation of a spikeless core particle: implications for capsid assembly
AUTHOR(S): Hazelton, Paul R.; Coombs, Kevin M.
CORPORATE SOURCE: Department of Medical Microbiology and Infectious Diseases, University of Manitoba, Winnipeg, MB, R3E 0W3, Can.
SOURCE: Journal of Virology (1999), 73(3), 2298-2308
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Previous studies which used intertypic **reassortants** of the wild-type **reovirus** serotype 1 Lang and the temperature-sensitive (ts) serotype 3 mutant clone tsA279 identified two ts lesions; one lesion, in the M2 gene segment, was associated with defective transmembrane transport of restrictively assembled virions (P. R. Hazelton and K. M. Coombs, Virol. 207:46-58, 1995). In the present study we show that the second lesion, in the L2 gene segment, which encodes the λ2 protein, is associated with the accumulation of a core-like particle defective for the λ2 pentameric spike. Physicochem., biochem., and immunol. studies showed that these structures were deficient for genomic double-stranded RNA, the core spike protein λ2, and the minor core protein μ2. Core particles with the λ2 spike structure accumulated after temperature shift-down from a restrictive to a permissive temperature in the presence of cycloheximide. These data suggest the spike-deficient, core-like particle is an assembly intermediate in reovirus morphogenesis. The existence of this naturally occurring primary core structure suggests that the core proteins λ1, λ3, and σ2 interact to initiate the process of virion capsid assembly through a dodecahedral mechanism. The next step in the proposed capsid assembly model would be the association of the minor core protein μ2, either preceding or collateral to the

condensation of the λ2 pentameric spike at the apices of the primary core structure. The assembly pathway of the reovirus double capsid is further elaborated when these observations are combined with structures identified in other studies.

REFERENCE COUNT: 109 THERE ARE 109 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 6 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1998:52905 CAPLUS
DOCUMENT NUMBER: 128:153025
TITLE: Reovirus induction of and sensitivity to beta interferon in cardiac myocyte cultures correlate with induction of myocarditis and are determined by viral core proteins
AUTHOR(S): Sherry, Barbara; Torres, Johann; Blum, Mary Ann
CORPORATE SOURCE: Dep. Microbiol., Pathol. Parasitol., Coll. Veterinary Med., North Carolina State Univ., Raleigh, NC, 27606, USA
SOURCE: Journal of Virology (1998), 72(2), 1314-1323
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Reovirus-induced acute myocarditis in mice serves as a model to investigate non-immune-mediated mechanisms of viral myocarditis. The authors have used primary cardiac myocyte cultures infected with a large panel of myocarditic and nonmyocarditic **reassortant reoviruses** to identify determinants of viral myocarditic potential. Here, they report that while both myocarditic and nonmyocarditic reoviruses kill cardiac myocytes, viral myocarditic potential correlates with viral spread through cardiac myocyte cultures and with cumulative cell death. To address the role of secreted interferon (IFN), the authors added anti-IFN-α/β antibody to infected cardiac myocyte cultures. Antibody benefited nonmyocarditic more than myocarditic virus spread, and this benefit was associated with the reovirus M1 and L2 genes. There was no benefit for a differentiated skeletal muscle cell line culture (C2C12 cells), suggesting cell type specificity. IFN-β induction in reovirus-infected cardiac myocyte cultures correlated with viral myocarditic potential and was associated with the reovirus M1, S2, and L2 genes. Sensitivity to the antiviral effects of IFN-α/β added to cardiac myocyte cultures also correlated with viral myocarditic potential and was associated with the same reovirus genes. Several reoviruses induced IFN-β levels discordant with their myocarditic phenotypes, and for those tested, sensitivity to IFN-α/β compensated for the anomalous induction levels. Thus, the combination of induction of and sensitivity to IFN-α/β is a determinant of reovirus myocarditic potential. Finally, a nonmyocarditic reovirus induced cardiac lesions in mice depleted of IFN-α/β, demonstrating that IFN-α/β is a determinant of reovirus-induced myocarditis. This provides the first identification of reovirus genes associated with IFN induction and sensitivity and provides the first evidence that IFN-β can be a determinant of viral myocarditis and reovirus disease.

REFERENCE COUNT: 72 THERE ARE 72 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 7 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1997:121570 CAPLUS
DOCUMENT NUMBER: 126:209335
TITLE: Mutations in type 3 reovirus that determine binding to sialic acid are contained in the fibrous tail domain of viral attachment protein σ1
AUTHOR(S): Chappell, James D.; Gunn, Veronica L.; Wetzel, J.

Denise; Baer, Geoffrey S.; Dermody, Terence S.
CORPORATE SOURCE: Department Microbiology and Immunology, Vanderbilt University School of Medicine, Nashville, TN, 37232, USA
SOURCE: Journal of Virology (1997), 71(3), 1834-1841
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The reovirus attachment protein, σ1, dets. numerous aspects of reovirus-induced disease, including viral virulence, pathways of spread, and tropism for certain types of cells in the central nervous system. The σ1 protein projects from the virion surface and consists of two distinct morphol. domains, a virion-distal globular domain known as the had and an elongated fibrous domain, termed the tail, which is anchored into the virion capsid. To better understand structure-function relationships of σ1 protein we conducted expts. to identify sequences in σ1 important for viral binding to sialic acid, a component of the receptor for type 3 reovirus. Three serotype 3 reovirus strains incapable of binding sialylated receptors were adapted to growth in murine erythroleukemia (MLE) cells, in which sialic acid is essential for reovirus infectivity. MEL-adapted (MA) mutant viruses isolated by serial passage in MEL cells acquired the capacity to bind sialic acid-containing receptors and demonstrated a dependence on sialic acid for infection of MEL cells. Anal. of **reassortant** viruses isolated from crosses of an MA mutant virus and a **reovirus** strain that does not bind sialic acid indicated that the σ1 protein is solely responsible for efficient growth of MA mutant viruses in MEL cells. The deduced σ1 amino acid sequences of the MA mutant viruses revealed that each strain contains a substitution within a short region of sequence in the σ1 tail predicted to form β-sheet. These studies identify specific sequences that determine the capacity of reovirus to bind sialylated receptors and suggest a location for a sialic acid-binding domain. Furthermore, the results support a model in which type 3 σ1 protein contains discrete receptor binding domains, one in the head and another in the tail that binds sialic acid.

REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 8 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1997:56469 CAPLUS
DOCUMENT NUMBER: 126:115544
TITLE: Reovirus variants selected during persistent infections of L cells contain mutations in the viral S1 and S4 genes and are altered in viral disassembly
AUTHOR(S): Wetzel, J. Denise; Wilson, Gregory J.; Baer, Geoffrey S.; Dunnigan, Lisa R.; Wright, Justin P.; Tang, David S. H.; Dermody, Terence S.
CORPORATE SOURCE: School of Medicine, Vanderbilt University, Nashville, TN, 37232, USA
SOURCE: Journal of Virology (1997), 71(2), 1362-1369
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Reoviruses isolated from persistently infected cultures (PI viruses) can grow in the presence of NH4Cl, a weak base that blocks acid-dependent proteolysis of viral outer-capsid proteins during viral entry into cells. **Reassortant** viruses isolated from crosses of wild-type (wt) **reovirus** strain, type 1 Lang, and 3 independent PI viruses, L/C, PI 2A1, and PI 3-1, were used to identify viral genes that segregate with the capacity of PI viruses to grow in cells treated with NH4Cl. Growth of reassortment viruses in NH4Cl-treated cells segregated with the S1 gene of L/C and the S4 gene of PI 2A1 and PI 3-1. The S1 gene encodes viral

attachment protein σ3. To identify mutations in σ3 selected during persistent reovirus infection, the S4 gene nucleotide sequences of L/C, PI 2A1, PI 3-1, and 4 addnl. PI viruses were determined The deduced amino acid sequences of σ3 protein of 6 of these PI viruses contained a tyrosine-to-histidine substitution at residue 354. To determine whether mutations selected during persistent infection alter cleavage of the viral outer capsid, the fate of viral structural proteins was assessed by SDS-PAGE after treatment of virions of wt and PI viruses with chymotrypsin in vitro. Proteolysis of PI virus outer-capsid proteins σ3 and μ1C occurred with faster kinetics than proteolysis of wt virus outer-capsid proteins. These results demonstrate that mutations in either the S1 or S4 gene alter acid-dependent disassembly of the reovirus outer capsid and suggest that increased efficiency of proteolysis of viral outer-capsid proteins is important for maintenance of persistent reovirus infections of cultured cells.

REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 9 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1996:626073 CAPLUS
DOCUMENT NUMBER: 125:270126
TITLE: Linkage between reovirus-induced apoptosis and inhibition of cellular DNA synthesis: role of the S1 and M2 genes
AUTHOR(S): Tyler, Kenneth L.; Squier, Margaret K. T.; Brown, Andrea L.; Pike, Bobbi; Willis, Derall; Oberhaus, Stephanie M.; Dermody, Terence S.; Cohen, J. John
CORPORATE SOURCE: Dep. Neurol., Univ. Colorado Health Sci. Cent., Neurol. Serv., Denver, CO, 80220, USA
SOURCE: Journal of Virology (1996), 70(11), 7984-7991
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The mammalian reoviruses are capable of inhibiting cellular DNA synthesis and inducing apoptosis. Reovirus strains type 3 Abney (T3A) and type 3 Dearing (T3D) inhibit cellular DNA synthesis and induce apoptosis to a substantially greater extent than strain type 1 Lang (T1L). T1L + T3A and T1L + T3D **reassortant** viruses were used to identify viral genes associated with differences in the capacities of **reovirus** strains to elicit these cellular responses to viral infection. The S1 and M2 genome segments determine differences in the capacities of both T1L + T3A and T1L + T3D reassortant viruses to inhibit cellular DNA synthesis and to induce apoptosis. These genes encode viral outer-capsid proteins that play important roles in viral attachment and disassembly. To extend these findings, field isolate strains of reovirus were used to determine whether the strain-specific differences in inhibition of cellular DNA synthesis and induction of apoptosis are also associated with viral serotype, a property determined by the S1 gene. In these expts., type 3 field isolate strains were found to inhibit cellular DNA synthesis and to induce apoptosis to a greater extent than type 1 field isolate strains. Statistical anal. of these data indicate a significant correlation between the capacity of T1L + T3A and T1L + T3D reassortant viruses and field isolate strains to inhibit cellular DNA synthesis and to induce apoptosis. These findings suggest that reovirus-induced inhibition of cellular DNA synthesis and induction of apoptosis are linked and that both phenomena are induced by early steps in the viral replication cycle.

L10 ANSWER 10 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1996:571053 CAPLUS
DOCUMENT NUMBER: 125:214166
TITLE: Nonrandom segregation of a parental alleles in

reovirus reassortants

AUTHOR(S): Nibert, Max. L.; Margraf, Rebecca L.; Coombs, Kevin M.
CORPORATE SOURCE: Inst. Mol. Virology, Univ. Wisconsin-Madison, Madison, WI, 53706, USA
SOURCE: Journal of Virology (1996), 70(10), 7295-7300
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB To test for nonrandom segregations among their 10 genomic RNA segments, the authors examined a set of 83 **reassortants** derived from mammalian **reovirus** type 1 Lang and type 3 Dearing. After confirming the genotypes of the reassortants, the authors performed statistical analyses on the distributions of parental alleles for each of the 10 gene segments, as well as for the 45 possible pairings of the 10 segments. The analyses revealed nonrandom assocns. of parental alleles in the L1-L2, L1-M1, L1-S1, and L3-S1 segment pairs, at levels indicating high statistical significance ($P < 0.005$). Such assocns. may reflect specific interactions between viral components (protein-protein, protein-RNA, or RNA-RNA) and may influence both the evolution of reoviruses in nature and their genetic anal. in the laboratory The data may also support an hypothesis that **reovirus reassortants** commonly contain mutations that improve their fitness for independent replication.

L10 ANSWER 11 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1996:570974 CAPLUS
DOCUMENT NUMBER: 125:270176
TITLE: Reovirus-induced acute myocarditis in mice correlates with viral RNA synthesis rather than generation of infectious virus in cardiac myocytes
AUTHOR(S): Sherry, Barbara; Baty, Catherine J.; Blum, Mary Ann
CORPORATE SOURCE: College Veterinary Medicine, North Carolina State University, Raleigh, NC, 27606, USA
SOURCE: Journal of Virology (1996), 70(10), 6709-6715
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The capacity for different **reovirus reassortant** viruses to induce acute myocarditis in mice correlates with cytopathogenic effect in primary cultures of murine cardiac myocytes. Multiple viral genes encoding proteins involved in viral RNA synthesis are determinants of this disease. The role of viral RNA synthesis in induction of acute myocarditis was therefore evaluated by infecting primary cultures of cardiac myocytes with a panel of myocarditic and nonmyocarditic viruses and quantitating RNA synthesis. RNA synthesis correlated with induction of myocarditis and with the S1 and M1 reovirus genes. Since one consequence of viral RNA synthesis is generation of infectious virus, viral yield from cardiac myocyte cultures was studied. Yield of infectious virus at an early time postinfection or as a final yield from primary infections did not correlate with myocarditis, but instead both correlated with the S1 gene. The S1 gene also determined the fraction of cells infected during primary infections in the culture, which varied dramatically between viruses. Viral yields per infected cell were similar for most myocarditic and nonmyocarditic reoviruses and did not correlate with induction of myocarditis or any reovirus gene. Together, the data provide 2 insights into reovirus-induced acute myocarditis in mice. First, while the S1 gene, which encodes the viral attachment protein σ1 (as well as a nonstructural protein, σ1s, of unknown function) does not determine the myocarditic potential of these viruses, it does determine the efficiency with which they infect cardiac myocytes. Second, while viral RNA synthesis is a determinant of acute myocarditis, this is not due to generation of infectious virus. This finding suggests that

some other consequence of viral RNA synthesis, for example, induction of interferon, may determine reovirus-induced acute myocarditis.

L10 ANSWER 12 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1996:13960 CAPLUS
DOCUMENT NUMBER: 124:108726
TITLE: Identification of signals required for the insertion of heterologous genome segments into the reovirus genome
AUTHOR(S): Roner, Michael R.; Lin, Peng-Nian; Nepleuv, Igor; Kong, Ling-Jie; Joklik, Wolfgang K.
CORPORATE SOURCE: Dep. Microbiol., Duke Univ. Med. Cent., Durham, NC, 27710, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (1995), 92(26), 12362-6
CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English

AB In cells simultaneously infected with any two of the three **reovirus** serotypes ST1, ST2, and ST3, up to 15% of the yields are intertypic **reassortants** that contain all possible combinations of parental genome segments. We have now found that not all genome segments in reassortants are wild type. In reassortants that possess more ST1 than ST3 genome segments, all ST1 genome segments appear to be wild type, but the incoming ST3 genome segments possess mutations that make them more similar to the ST1 genome segments that they replace. In reassortants resulting from crosses of the more distantly related ST3 and ST2 viruses that possess a majority of ST3 genome segments, all incoming ST2 genome segments are wild type, but the ST3 S4 genome segment possesses two mutations, G74 to A and G624 to A, that function as acceptance signals. Recognition of these signals has far-reaching implications for the construction of reoviruses with novel properties and functions.

L10 ANSWER 13 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1995:990528 CAPLUS
DOCUMENT NUMBER: 124:50503
TITLE: Role of the μ1 protein in reovirus stability and capacity to cause chromium release from host cells
AUTHOR(S): Hooper, Jay W.; Fields, Bernard N.
CORPORATE SOURCE: Dep. Microbiol. Mol. Genet., Harvard Med. Sch., Boston, MA, 02115, USA
SOURCE: Journal of Virology (1996), 70(1), 459-67
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The reovirus M2 gene is associated with the capacity of type 3 strain Abney (T3A) intermediate subviral particles (ISVPs) to permeabilize cell membranes as measured by 51Cr release. In addition, reovirus mutants with lesions in the M2 gene can be selected by heating the virus at 37° for 20 min in 33% EtOH. In this report, the mechanism by which the reovirus M2 gene product (the μ1 protein) influences the capacity of reovirus ISVPs to permeabilize membranes was investigated using EtOH-decreased capacity to cause 51Cr release relative to that of wild-type T3A. Sequence anal. of the M2 genes of wild-type T3A and T3A mutants indicated that each mutant possesses a single amino acid substitution in a central region of the 708-amino-acid μ1 protein: JH2 (residue 466, Tyr to Cys), JH3 (residue 459, Lys to Glu), and JH4 (residue 497 Pro to Ser). Assays performed with **reovirus** natural isolates, **reassortants**, and a set of previously characterized type 3 strain Dearing (T3D) EtOH-resistant mutants revealed a strong correlation between EtOH sensitivity and the capacity to cause 51Cr release. ISVPs generated from the T3A and T3D mutants were stable when

heated to 50°, whereas wild-type T3A ISVPs are inactivated under these conditions. Together, these data suggest that amino acid substitutions in a central region of the μ1 protein affect the capacity of the ISVP to permeabilize L-cell membranes by altering the stability of the virus particle.

L10 ANSWER 14 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1995:410152 CAPLUS
DOCUMENT NUMBER: 122:206187
TITLE: What reassorts when reovirus genome segments reassort?
AUTHOR(S): Joklik, Wolfgang K.; Roner, Michael R.
CORPORATE SOURCE: Dep. Microbiol., Duke Univ. Med. Cent., Durham, NC, 27710, USA
SOURCE: Journal of Biological Chemistry (1995), 270(9), 4181-4
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 43 refs. Topics discussed include packaging of the 3 single-stranded ϕ6 genome segment precursors, the structure of **reovirus** RNA assortment complexes, the infectious **reovirus** RNA system, the nature of the genome segments in **reassortants**, and significance of mutations in **reovirus reassortants**
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L10 ANSWER 15 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1995:374535 CAPLUS
DOCUMENT NUMBER: 122:157573
TITLE: Reovirus mutant tsA279 has temperature-sensitive lesions in the M2 and L2 genes and association of M2 gene with decreased viral protein production and blockage in transmembrane transport
AUTHOR(S): Hazelton, Paul R.; Coombs, Kevin M.
CORPORATE SOURCE: Dep. Med. Microbiol. Infectious Diseases, Univ. Manitoba, Winnipeg, MB, R3E 0W3, Can.
SOURCE: Virology (1995), 207(1), 46-58
CODEN: VIRLAX; ISSN: 0042-6822
PUBLISHER: Academic
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Temperature-sensitive mutants provide an ideal means for dissecting viral assembly pathways. The morphol. variants produced by and biol. characteristics of tsA279, a previously uncharacterized mutant from the Fields' panel of temperature-sensitive mutants of reovirus, were determined under restrictive growth conditions. The mutant showed a distinctive pattern of increased temperature sensitivity as the temperature was raised from 39° to 40°. Wild-type **reovirus** type 1 Lang and the mutant were crossed to generate **reassortants**. Efficiency of plating analyses of the reassortants showed that tsA279 has temperature-sensitive lesions in two genes, a mildly temperature-sensitive one in L2, which encodes core spike protein λ2, and a stronger, dominant lesion in M2, which encodes major outer capsid protein μ1. Electron microscopic examination of thin-sectioned tsA279-infected cells showed three ways in which the mutant phenotypes were expressed. The mutant appeared to be blocked in transmembrane transport of virions, a phenotype that mapped to the M2 gene; the mutant produced significantly reduced amts. of identifiable particles; and those particles that were produced appeared to be morphol. variants. Immunofluorescent microscopy and immunopptns. of tsA279- and various T1L x tsA279 reassortant-infected cells suggested that the reduction in observed progeny was caused by a decreased production of viral proteins at the nonpermissive temperature This phenotype also mapped to the mutant M2 gene.

L10 ANSWER 16 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1995:271862 CAPLUS
DOCUMENT NUMBER: 122:48211
TITLE: Genetic mapping of reovirus virulence and organ tropism in severe combined immunodeficient mice: organ-specific virulence genes
AUTHOR(S): Haller, Barbara L.; Barkon, Melissa L.; Vogler, George P.; Virgin, Herbert W., IV
CORPORATE SOURCE: Cent. Immunology, Washington Univ. Sch. Med., St. Louis, MO, 63110, USA
SOURCE: Journal of Virology (1995), 69(1), 357-64
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We used **reovirus reassortant** genetics and severe combined immunodeficient (SCID) mice to define viral genes important for organ tropism and virulence in the absence of antigen-specific immunity. Adult SCID mice infected with reovirus serotype 1 strain Lang (T1L) died after 20 ± 6 days, while infection with serotype 3 strain Dearing (T3D) was lethal after 77 ± 22 days. One hundred forty-five adult SCID mice were infected with T1L, T3D, and 25 different T1L + T3D **reassortant reoviruses**, and gene segments associated with the increased virulence of T1L were identified. Gene segments S1, L2, M1, and L1 account for >90% of the genetically determined increase in T1L virulence. Gene segment M1 was independently important for virulence, with S1, L2, and L1 alone or in combination also playing a role. T1L grew to higher titers in multiple organs and caused more severe hepatitis than T3D. Seventy adult SCID mice, T1L, T3D, and 15 T1L + T3D reassortant viruses were used to map genetic determinants of viral titers in the brain, intestines, and liver, as well as the severity of hepatitis. Different sets of gene segments were important for determining viral titers in different organs. Gene segments L1 (encoding a core protein) and L2 (encoding the core spike of the virion) were important in all of the organs analyzed. The M1 gene segment (encoding a core protein), but not the S1 gene segment, was a critical determinant of reovirus titer in the liver and severity of hepatitis. The S1 gene segment (encoding the viral cell attachment protein and a nonstructural protein), but not the M1 gene segment, was a critical determinant of titers in intestines and brains. These studies demonstrate that viral growth in different organs is dependent on different subsets of the genes important for virulence. The virion-associated protein products of the four gene segments (L1, L2, M1, and S1) important for virulence and organ tropism in SCID mice likely form a structural unit, the reovirus vertex. Organs (the brain and intestines vs. the liver) differ in properties that determine which virulence genes, and thus which parts of this structural unit, are important.

L10 ANSWER 17 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1994:101716 CAPLUS
DOCUMENT NUMBER: 120:101716
TITLE: Studies of the major reovirus core protein σ2: reversion of the assembly-defective mutant tsC447 is an intragenic process and involves back mutation of Asp-383 to Asn
AUTHOR(S): Coombs, Kevin M.; Mak, Sin Chi; Petrycky-Cox, Lydia D.
CORPORATE SOURCE: Dep. Med. Microbiol. Infect. Dis., Univ. Manitoba, Winnipeg, MB, R3E 0W3, Can.
SOURCE: Journal of Virology (1994), 68(1), 177-86
CODEN: JOVIAM; ISSN: 0022-538X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The reovirus group C temperature-sensitive mutant tsC447, whose defect maps to the S2 gene, which encodes the major core protein σ2, fails to

assemble core particles at the nonpermissive temperature To identify other proteins that may interact with σ2 during assembly, the authors generated and examined 10 independent revertants of the mutant. To determine which gene(s) carried a compensatory suppressor mutation(s), the authors generated intertypic **reassortants** between wild-type **reovirus** serotype 1 Lang and each revertant and determined the temperature sensitivities of the **reassortants** by efficiency-of-plating assays. Results of the efficiency-of-plating analyses indicated that reversion of the tsC447 defect was an intragenic process in all revertants. To identify the region(s) of σ2 that had reverted, the authors determined the nucleotide sequences of the S2 genes. In all revertant sequences examined, the G at nucleotide position 1166 in tsC447 had reverted to the A present in the wild-type sequence. This reversion leads to the restoration of a wild-type asparagine (in place of a mutant aspartic acid) at amino acid 383 in the σ2 sequence. These results collectively indicate that the functional lesion in tsC447 is Asp-383 and that this lesion cannot be corrected by alterations in other core proteins. These observations suggest that this region of σ2, which may be important in mediating assembly of the core particle, does not interact significantly with other reovirus proteins.

L10 ANSWER 18 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1993:33859 CAPLUS
DOCUMENT NUMBER: 118:33859
TITLE: Identification of sequence elements containing signals for replication and encapsidation of the reovirus M1 genome segment
AUTHOR(S): Zou, S.; Brown, E. G.
CORPORATE SOURCE: Fac. Med., Univ. Ottawa, Ottawa, ON, K1H 8M5, Can.
SOURCE: Virology (1992), 186(2), 377-88
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In reovirus the genetic signals that control genome replication and encapsidation are unknown. Serial passage of reovirus results in the accumulation of deletion mutants that contain fragments of genome segments. The smallest fragments found in deletion mutants will consist of the min. essential sequences for genome replication and assembly. T1 + T3 reassortants containing the L2 segment from T3 and the M3 segment derived from T1 generate deletions in segment M1 on serial passage. Fragments of M1 segments were produced by serial passage, characterized by PAGE and Northern blotting before amplification by PCR, cloning, and sequencing. Thirteen of the smallest deletion fragments were sequenced. All of the smallest fragments contained sequences from both termini of segment M1. The smallest fragment was 344 nucleotides long. The consensus sequences consisted of 132-135 nucleotides from the 5' end of the plus strand and 183-185 nucleotides from the 3' end of the plus strand. It is concluded that these regions contain all the signals necessary for the replication and assembly of the M1 genome segment.

L10 ANSWER 19 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1990:510135 CAPLUS
DOCUMENT NUMBER: 113:110135
TITLE: Selection of genome segments following coinfection of chicken fibroblasts with avian reoviruses
AUTHOR(S): Ni, Yawei; Kemp, Maurice C.
CORPORATE SOURCE: Coll. Vet. Med., Texas A and M Univ., College Station, TX, 77843-4467, USA
SOURCE: Virology (1990), 177(2), 625-33
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Two avian **reoviruses** (883 and 176) shown to have distinct growth kinetics were used to coinfect chicken embryonic fibroblasts

asynchronously to generate **reassortants**. More than 300 plaque-derived clones were obtained from passage 3 of two sep. coinfections made at different m.o.i. and time intervals between infection and superinfection. The genome electropherotype of each plaque-derived clone was determined, and a diverse group of reassortants were detected. Genome segments 883 M2 and 176 S1 were shown to be preferentially selected. The preferential selection of the 176 S1 segment was shown to be a virus growth-determined nonrandom event conferred by the function of 176 S1 segment, whereas the data suggest that a factor(s) other than viral growth properties was involved in the preferential selection of 883 M2 segment.

L10 ANSWER 20 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1990:52033 CAPLUS
DOCUMENT NUMBER: 112:52033
TITLE: The function of reovirus proteins during the reovirus multiplication cycle: analysis using monoreassortants
AUTHOR(S): Moody, Mark D.; Joklik, Wolfgang K.
CORPORATE SOURCE: Med. Cent., Duke Univ., Durham, NC, 27710, USA
SOURCE: Virology (1989), 173(2), 437-46
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English
AB When cultured cells are injected with mixts. of cores of two **reovirus** strains, many **reassortants** are monoreassortants, i.e., virus particles that contain one genome segment of 1 parent and 9 genome segments of the other. The authors isolated two complete sets of monoreassortants, those that contain a single serotype 2 genome segment and 9 serotype 3 genome segments, and those that contain 1 serotype 3 genome segment and 9 serotype 1 genome segments. The former set of monoreassortants (because reovirus serotypes 2 and 3 are less closely related than serotypes 1 and 3) was used to assess the effect of all 10 genome segments, or rather of the proteins that they encode, in controlling parameters of the reovirus multiplication cycle such as yield size, extent of viral ssRNA, dsRNA and protein synthesis, plaque size, and cytopathogenicity. Among the major findings are: proteins λ2, μ1/μ1C, and σ3 control yield size and extent of RNA and protein synthesis; proteins μ2 and σ1 control severity of cytopathic effects; and proteins σ1, μ1/μ1C, and μ2 control plaque size. Identification of monoreassortant phenotypes is useful for identifying which viral proteins are functionally involved at the various stages of the reovirus multiplication cycle.

L10 ANSWER 21 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1990:1542 CAPLUS
DOCUMENT NUMBER: 112:1542
TITLE: The reovirus M1 gene, encoding a viral core protein, is associated with the myocarditic phenotype of a reovirus variant
AUTHOR(S): Sherry, Barbara; Fields, Bernard N.
CORPORATE SOURCE: Dep. Microbiol. Mol. Genet., Harvard Med. Sch., Boston, MA, 02115, USA
SOURCE: Journal of Virology (1989), 63(11), 4850-6
CODEN: JOVIAM; ISSN: 0022-538X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Reoviruses contain a genome composed of 10 double-stranded RNA gene segments. A **reovirus reassortant**, 8B, derived from type 1 Lang (T1L) and type 3 Dearing (T3D), displayed a phenotype unlike that of either of its parents in that it efficiently induced numerous macroscopic external cardiac lesions in neonatal mice. A panel of T1L/T3D reassortants and a panel of reassortants derived from 8B were used to determine whether novel T1L/T3D gene assocns. in 8B were responsible for its myocarditic phenotype. The results eliminated the possibility that any

T1L/T3D gene combination found in 8B, from 2 genes to all 10 genes, was the explanation for its induction of cardiac lesions. This suggested that a mutation(s) in an 8B gene(s) might be responsible for induction of the myocarditis. Statistical anal. of expts. with 31 reassortants derived from 8B revealed a highly significant association of the 8B M1 gene with induction of cardiac lesions. The reovirus M1 gene encodes a viral core protein of unknown function, although evidence suggests a potential role in core structure and/or viral RNA synthesis. This represents the first report of the association of a viral gene with induction of myocarditis.

L10 ANSWER 22 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1989:167207 CAPLUS
DOCUMENT NUMBER: 110:167207
TITLE: Growth and survival of reovirus in intestinal tissue: role of the L2 and S1 genes
AUTHOR(S): Bodkin, Dinah K.; Fields, Bernard N.
CORPORATE SOURCE: Dep. Microbiol. Mol. Genet., Harvard Med. Sch., Boston, MA, 02115, USA
SOURCE: Journal of Virology (1989), 63(3), 1188-93
CODEN: JOVIAM; ISSN: 0022-538X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Reovirus serotype 1 Lang can be recovered in high titer from the intestines of neonatal mice up to day 8 after peroral inoculation. By contrast, reovirus serotype 3 Dearing cannot be recovered from intestinal tissue past day 4 after peroral inoculation. This difference between the 2 **reoviruses** was mapped by using **reassortants** generated from nonmutagenized laboratory stocks. When the L2 and S1 genes of **reovirus** serotype 3 Daring were present in **reassortants**, the **reassortants** behaved like serotype 3 Dearing in exhibiting a decreased capacity to be recovered from intestinal tissue. Likewise, viruses which contained the L2 and S2 genes from serotype 1 Land exhibited an enhanced capacity to grow and survive, which is characteristic of serotype 1 Lang. Thus, the capacity of reovirus to survive in intestinal tissue was determined by the L2 and S1 genes.

L10 ANSWER 23 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1987:420639 CAPLUS
DOCUMENT NUMBER: 107:20639
TITLE: Inhibition of reovirus type 3 binding to host cells by sialylated glycoproteins is mediated through the viral attachment protein
AUTHOR(S): Pacitti, Anne F.; Gentsch, Jon R.
CORPORATE SOURCE: Sch. Med., Univ. Pennsylvania, Philadelphia, PA, 19104-6076, USA
SOURCE: Journal of Virology (1987), 61(5), 1407-15
CODEN: JOVIAM; ISSN: 0022-538X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The interaction of mammalian reoviruses with sialylated glycoproteins was studied and found to be highly serotype specific in that attachment of type 3 Dearing reovirus to murine L cell receptors could be strongly inhibited by bovine submaxillary mucin (BSM), fetuin, and alpha1 acid glycoprotein, albeit at different efficiencies, whereas attachment of type 1 Lang reovirus was inhibited only by fetuin. It was subsequently demonstrated, by using **reassortants** between type 3 and 1 **reoviruses**, that inhibition of **reovirus** attachment to cell receptors was specified by the viral attachment protein gene S1. Using a solid-phase binding assay, it was further demonstrated that the ability of **reovirus** type 3 or **reassortant** 1HA3 and the inability of **reovirus** type 1 or **reassortant** 3HA1 to bind avidly to BSM was a property of the viral S1 genome segment and required the presence of sialic acid residues on BSM oligosaccharides. Taken together, these results demonstrated that there is a

serotype-specific difference in the ability of the reovirus attachment protein, sigma 1, to interact with sialylated oligosaccharides of glycoproteins. The interaction of reovirus type 3 with siaylated oligosaccharides of BSM is dramatically affected by the degree of O-acetylation of their sialic acid residues, as indicated by the findings that chemical removal of O-acetyl groups stimulated reovirus type 3 attachment to BSM, whereas preferential removal of residues lacking or possessing reduced amts. of O-acetyl groups per sialic acid mol. with Vibrio cholerae silidase abolished binding. BSM was 10 times more potent in inhibiting attachment of infectious reovirus to L cells than was V. cholerae-treated BSM. The results are consistent with the hypothesis that sialylated oligosaccharides on host cells or erythrocytes may act as binding sites or components of binding sites for type 3 reovirus through a specific interaction with the virus attachment protein.

L10 ANSWER 24 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1986:509708 CAPLUS
DOCUMENT NUMBER: 105:109708
TITLE: Distinct pathways of viral spread in the host determined by reovirus S1 gene segment
AUTHOR(S): Tyler, Kenneth L.; McPhee, Dale A.; Fields, Bernard N.
CORPORATE SOURCE: Dep. Microbiol. Mol. Genet., Harvard Med. Sch., Boston, MA, 02115, USA
SOURCE: Science (Washington, DC, United States) (1986), 233(4765), 770-4
CODEN: SCIEAS; ISSN: 0036-8075
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The genetic and mol. mechanisms that determine the capacity of a virus to utilize distinct pathways of spread in an infected host were examined by using reoviruses. Both reovirus type 1 and reovirus type 3 spread to the spinal cord following inoculation into the hindlimb or forelimb footpad of newborn mice. For type 3, this spread is through nerves and occurs via the microtubule-associated system of fast axonal transport. By contrast, type 1 spreads to the spinal cord through the bloodstream. With the use of **reassortant** viruses containing various combinations of double-stranded RNA segments (genes) derived from type 1 and type 3, the viral S1 double-stranded RNA segment was shown to be responsible for determining the capacity of **reoviruses** to spread to the central nervous system through these distinct pathways.

L10 ANSWER 25 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1985:592998 CAPLUS
DOCUMENT NUMBER: 103:192998
TITLE: Genetic reassortment of mammalian reoviruses in mice
AUTHOR(S): Wenske, Elizabeth A.; Chanock, Stephen J.; Krata, Lewis; Fields, Bernard N.
CORPORATE SOURCE: Dep. Microbiol. Mol. Genet., Harvard Med. Sch., Boston, MA, 02115, USA
SOURCE: Journal of Virology (1985), 56(2), 613-16
CODEN: JOVIAM; ISSN: 0022-538X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Reassortants** between type 1 (Lang) and type 3 (Dearing) **reoviruses** were isolated from suckling mice infected perorally with an inoculum containing both type 1 and type 3 viruses. A total of 5 distinct reassortants (designated as E1 through E5) were isolated from animals during the course of the experiment Two reassortants (E1 and E2) represented the majority of the reassortants isolated. The majority of genes of types E1 and E2 were derived from type 1 (Lang). However, E1 had an M2 gene and an S1 gene derived from type 3 (Dearing), whereas E2 had M2 and S2 genes derived from type 3 (Dearing). Thus, nonrandom reassortment between mammalian reoviruses can be demonstrated in vivo.

L10 ANSWER 26 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1984:526598 CAPLUS
DOCUMENT NUMBER: 101:126598
TITLE: Extragenic suppression of temperature-sensitive phenotype in reovirus: mapping suppressor mutations
AUTHOR(S): McPhillips, Thomas H.; Ramig, Robert F.
CORPORATE SOURCE: Texas Med. Cent., Baylor Coll. Med., Houston, TX, 77030, USA
SOURCE: Virology (1984), 135(2), 428-39
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Independently isolated, spontaneous pseudorevertants of temperature-sensitive (ts) mutants of reovirus type 3 have previously been genetically characterized (R. F. Ramig and B. N. Fields, 1979). Eighteen of these pseudorevertants were backcrossed to wild-type **reovirus** type 1 and **reassortant** progeny expressing the parental ts phenotype were selected. Anal. of segregation of genome segments in the reassortant, parental ts, progeny cloned allowed the determination of the genome segment bearing the suppressor mutation of four pseudorevertants. The suppressor of tsA(201) phenotype mapped to segment S4 in the pseudorevertants RtsA(201)101 and RtsA(201)121 and to segment L3 in pseudorevertant RtsA(201)122. The suppressor of tsB(352) phenotype mapped to segment S1 in the pseudorevertant RtsB(352)b. In two other pseudorevertants the suppressor could not be mapped to a single genome segment due to the small number of progeny clones examined These genetic results indirectly support the compensating protein interactions hypothesis for the mechanism of suppression.

L10 ANSWER 27 OF 27 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1983:105292 CAPLUS
DOCUMENT NUMBER: 98:105292
TITLE: The σ1 protein determines the extent of spread of reovirus from the gastrointestinal tract of mice
AUTHOR(S): Kauffman, Robert S.; Wolf, Jacqueline L.; Finberg, Robert; Trier, Jerry S.; Fields, Bernard N.
CORPORATE SOURCE: Dep. Microbiol., Harvard Med. Sch., Boston, MA, 02115, USA
SOURCE: Virology (1983), 124(2), 403-10
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English
AB After intragastric inoculation of adult mice, type 1 reovirus was initially concentrated in Peyer's patches over the first 4 h after inoculation, then spread sequentially to the mesenteric lymph nodes and spleen. For type 3 reovirus, however, initial entry into Peyer's patches in adult mice was followed by loss of viral infectivity so that by 4 h after inoculation virtually no infections virus was detected in the intestine, and spread to extraintestinal tissues did not occur. In 10-day-old mice, type 3 was capable of spread to the mesenteric lymph nodes but not the spleen. Thus, as animals aged there was a greater restriction of the spread of type 3 from the intestine. Studies using a field isolate of type 3 **reovirus** that is resistant to intestinal proteases, and genetic studies utilizing type 1 X type 3 viral **reassortants**, revealed that the viral σ1 protein determined the capacity of **reovirus** to spread from the intestine in both adult and 10-day-old mice. Thus, the interaction of reovirus with host defense mechanisms, and the age-dependent restriction of spread of type 3 reovirus from the intestine are mediated by the viral σ1 protein.

=> L10 and growth ability

```
       1172515 GROWTH
          4150 GROWTHS
       1174646 GROWTH
                 (GROWTH OR GROWTHS)
        367939 ABILITY
         17360 ABILITIES
        381240 ABILITY
                 (ABILITY OR ABILITIES)
           257 GROWTH ABILITY
                 (GROWTH(W)ABILITY)
L11          0 L10 AND GROWTH ABILITY

=> growth and L10
       1172515 GROWTH
          4150 GROWTHS
       1174646 GROWTH
                 (GROWTH OR GROWTHS)
L12          6 GROWTH AND L10

=> D L12 IBIB ABS 1-6

L12  ANSWER 1 OF 6  CAPLUS  COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER:         1997:121570  CAPLUS
DOCUMENT NUMBER:          126:209335
TITLE:                    Mutations in type 3 reovirus that determine binding to
                          sialic acid are contained in the fibrous tail domain
                          of viral attachment protein σ1
AUTHOR(S):                Chappell, James D.; Gunn, Veronica L.; Wetzel, J.
                          Denise; Baer, Geoffrey S.; Dermody, Terence S.
CORPORATE SOURCE:         Department Microbiology and Immunology, Vanderbilt
                          University School of Medicine, Nashville, TN, 37232,
                          USA
SOURCE:                   Journal of Virology (1997), 71(3), 1834-1841
                          CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER:                American Society for Microbiology
DOCUMENT TYPE:            Journal
LANGUAGE:                 English
AB   The reovirus attachment protein, σ1, dets. numerous aspects of
     reovirus-induced disease, including viral virulence, pathways of spread,
     and tropism for certain types of cells in the central nervous system.  The
     σ1 protein projects from the virion surface and consists of two
     distinct morphol. domains, a virion-distal globular domain known as the
     had and an elongated fibrous domain, termed the tail, which is anchored
     into the virion capsid.  To better understand structure-function
     relationships of σ1 protein we conducted expts. to identify
     sequences in σ1 important for viral binding to sialic acid, a
     component of the receptor for type 3 reovirus.  Three serotype 3 reovirus
     strains incapable of binding sialylated receptors were adapted to
     growth in murine erythroleukemia (MLE) cells, in which sialic acid
     is essential for reovirus infectivity.  MEL-adapted (MA) mutant viruses
     isolated by serial passage in MEL cells acquired the capacity to bind
     sialic acid-containing receptors and demonstrated a dependence on sialic acid
     for infection of MEL cells.  Anal. of reassortant viruses
     isolated from crosses of an MA mutant virus and a reovirus
     strain that does not bind sialic acid indicated that the σ1 protein
     is solely responsible for efficient growth of MA mutant viruses
     in MEL cells.  The deduced σ1 amino acid sequences of the MA mutant
     viruses revealed that each strain contains a substitution within a short
     region of sequence in the σ1 tail predicted to form β-sheet.
     These studies identify specific sequences that determine the capacity of
     reovirus to bind sialylated receptors and suggest a location for a sialic
     acid-binding domain.  Furthermore, the results support a model in which
     type 3 σ1 protein contains discrete receptor binding domains, one in
     the head and another in the tail that binds sialic acid.
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REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1997:56469 CAPLUS
DOCUMENT NUMBER: 126:115544
TITLE: Reovirus variants selected during persistent infections of L cells contain mutations in the viral S1 and S4 genes and are altered in viral disassembly
AUTHOR(S): Wetzel, J. Denise; Wilson, Gregory J.; Baer, Geoffrey S.; Dunnigan, Lisa R.; Wright, Justin P.; Tang, David S. H.; Dermody, Terence S.
CORPORATE SOURCE: School of Medicine, Vanderbilt University, Nashville, TN, 37232, USA
SOURCE: Journal of Virology (1997), 71(2), 1362-1369
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Reoviruses isolated from persistently infected cultures (PI viruses) can grow in the presence of NH4Cl, a weak base that blocks acid-dependent proteolysis of viral outer-capsid proteins during viral entry into cells. **Reassortant** viruses isolated from crosses of wild-type (wt) **reovirus** strain, type 1 Lang, and 3 independent PI viruses, L/C, PI 2A1, and PI 3-1, were used to identify viral genes that segregate with the capacity of PI viruses to grow in cells treated with NH4Cl. **Growth** of reassortment viruses in NH4Cl-treated cells segregated with the S1 gene of L/C and the S4 gene of PI 2A1 and PI 3-1. The S1 gene encodes viral attachment protein σ3. To identify mutations in σ3 selected during persistent reovirus infection, the S4 gene nucleotide sequences of L/C, PI 2A1, PI 3-1, and 4 addnl. PI viruses were determined The deduced amino acid sequences of σ3 protein of 6 of these PI viruses contained a tyrosine-to-histidine substitution at residue 354. To determine whether mutations selected during persistent infection alter cleavage of the viral outer capsid, the fate of viral structural proteins was assessed by SDS-PAGE after treatment of virions of wt and PI viruses with chymotrypsin in vitro. Proteolysis of PI virus outer-capsid proteins σ3 and μ1C occurred with faster kinetics than proteolysis of wt virus outer-capsid proteins. These results demonstrate that mutations in either the S1 or S4 gene alter acid-dependent disassembly of the reovirus outer capsid and suggest that increased efficiency of proteolysis of viral outer-capsid proteins is important for maintenance of persistent reovirus infections of cultured cells.
REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1995:374535 CAPLUS
DOCUMENT NUMBER: 122:157573
TITLE: Reovirus mutant tsA279 has temperature-sensitive lesions in the M2 and L2 genes and association of M2 gene with decreased viral protein production and blockage in transmembrane transport
AUTHOR(S): Hazelton, Paul R.; Coombs, Kevin M.
CORPORATE SOURCE: Dep. Med. Microbiol. Infectious Diseases, Univ. Manitoba, Winnipeg, MB, R3E 0W3, Can.
SOURCE: Virology (1995), 207(1), 46-58
CODEN: VIRLAX; ISSN: 0042-6822
PUBLISHER: Academic
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Temperature-sensitive mutants provide an ideal means for dissecting viral assembly pathways. The morphol. variants produced by and biol. characteristics of tsA279, a previously uncharacterized mutant from the

Fields' panel of temperature-sensitive mutants of reovirus, were determined under restrictive **growth** conditions. The mutant showed a distinctive pattern of increased temperature sensitivity as the temperature was raised from 39° to 40°. Wild-type **reovirus** type 1 Lang and the mutant were crossed to generate **reassortants**. Efficiency of plating analyses of the reassortants showed that tsA279 has temperature-sensitive lesions in two genes, a mildly temperature-sensitive one in L2, which encodes core spike protein λ2, and a stronger, dominant lesion in M2, which encodes major outer capsid protein μ1. Electron microscopic examination of thin-sectioned tsA279-infected cells showed three ways in which the mutant phenotypes were expressed. The mutant appeared to be blocked in transmembrane transport of virions, a phenotype that mapped to the M2 gene; the mutant produced significantly reduced amts. of identifiable particles; and those particles that were produced appeared to be morphol. variants. Immunofluorescent microscopy and immunopptns. of tsA279- and various T1L x tsA279 reassortant-infected cells suggested that the reduction in observed progeny was caused by a decreased production of viral proteins at the nonpermissive temperature This phenotype also mapped to the mutant M2 gene.

L12 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1995:271862 CAPLUS
DOCUMENT NUMBER: 122:48211
TITLE: Genetic mapping of reovirus virulence and organ tropism in severe combined immunodeficient mice: organ-specific virulence genes
AUTHOR(S): Haller, Barbara L.; Barkon, Melissa L.; Vogler, George P.; Virgin, Herbert W., IV
CORPORATE SOURCE: Cent. Immunology, Washington Univ. Sch. Med., St. Louis, MO, 63110, USA
SOURCE: Journal of Virology (1995), 69(1), 357-64
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We used **reovirus reassortant** genetics and severe combined immunodeficient (SCID) mice to define viral genes important for organ tropism and virulence in the absence of antigen-specific immunity. Adult SCID mice infected with reovirus serotype 1 strain Lang (T1L) died after 20 ± 6 days, while infection with serotype 3 strain Dearing (T3D) was lethal after 77 ± 22 days. One hundred forty-five adult SCID mice were infected with T1L, T3D, and 25 different T1L + T3D **reassortant reoviruses**, and gene segments associated with the increased virulence of T1L were identified. Gene segments S1, L2, M1, and L1 account for >90% of the genetically determined increase in T1L virulence. Gene segment M1 was independently important for virulence, with S1, L2, and L1 alone or in combination also playing a role. T1L grew to higher titers in multiple organs and caused more severe hepatitis than T3D. Seventy adult SCID mice, T1L, T3D, and 15 T1L + T3D reassortant viruses were used to map genetic determinants of viral titers in the brain, intestines, and liver, as well as the severity of hepatitis. Different sets of gene segments were important for determining viral titers in different organs. Gene segments L1 (encoding a core protein) and L2 (encoding the core spike of the virion) were important in all of the organs analyzed. The M1 gene segment (encoding a core protein), but not the S1 gene segment, was a critical determinant of reovirus titer in the liver and severity of hepatitis. The S1 gene segment (encoding the viral cell attachment protein and a nonstructural protein), but not the M1 gene segment, was a critical determinant of titers in intestines and brains. These studies demonstrate that viral **growth** in different organs is dependent on different subsets of the genes important for virulence. The virion-associated protein products of the four gene segments (L1, L2, M1,

and S1) important for virulence and organ tropism in SCID mice likely form a structural unit, the reovirus vertex. Organs (the brain and intestines vs. the liver) differ in properties that determine which virulence genes, and thus which parts of this structural unit, are important.

L12 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1990:510135 CAPLUS
DOCUMENT NUMBER: 113:110135
TITLE: Selection of genome segments following coinfection of chicken fibroblasts with avian reoviruses
AUTHOR(S): Ni, Yawei; Kemp, Maurice C.
CORPORATE SOURCE: Coll. Vet. Med., Texas A and M Univ., College Station, TX, 77843-4467, USA
SOURCE: Virology (1990), 177(2), 625-33
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Two avian **reoviruses** (883 and 176) shown to have distinct **growth** kinetics were used to coinfect chicken embryonic fibroblasts asynchronously to generate **reassortants**. More than 300 plaque-derived clones were obtained from passage 3 of two sep. coinfections made at different m.o.i. and time intervals between infection and superinfection. The genome electropherotype of each plaque-derived clone was determined, and a diverse group of reassortants were detected. Genome segments 883 M2 and 176 S1 were shown to be preferentially selected. The preferential selection of the 176 S1 segment was shown to be a virus **growth**-determined nonrandom event conferred by the function of 176 S1 segment, whereas the data suggest that a factor(s) other than viral **growth** properties was involved in the preferential selection of 883 M2 segment.

L12 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1989:167207 CAPLUS
DOCUMENT NUMBER: 110:167207
TITLE: **Growth** and survival of reovirus in intestinal tissue: role of the L2 and S1 genes
AUTHOR(S): Bodkin, Dinah K.; Fields, Bernard N.
CORPORATE SOURCE: Dep. Microbiol. Mol. Genet., Harvard Med. Sch., Boston, MA, 02115, USA
SOURCE: Journal of Virology (1989), 63(3), 1188-93
CODEN: JOVIAM; ISSN: 0022-538X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Reovirus serotype 1 Lang can be recovered in high titer from the intestines of neonatal mice up to day 8 after peroral inoculation. By contrast, reovirus serotype 3 Dearing cannot be recovered from intestinal tissue past day 4 after peroral inoculation. This difference between the 2 **reoviruses** was mapped by using **reassortants** generated from nonmutagenized laboratory stocks. When the L2 and S1 genes of **reovirus** serotype 3 Daring were present in **reassortants**, the **reassortants** behaved like serotype 3 Dearing in exhibiting a decreased capacity to be recovered from intestinal tissue. Likewise, viruses which contained the L2 and S2 genes from serotype 1 Land exhibited an enhanced capacity to grow and survive, which is characteristic of serotype 1 Lang. Thus, the capacity of reovirus to survive in intestinal tissue was determined by the L2 and S1 genes.

=> HEK and reovirus
4336 HEK
18 HEKS
4344 HEK
(HEK OR HEKS)
1881 REOVIRUS

313 REOVIRUSES
1946 REOVIRUS
(REOVIRUS OR REOVIRUSES)
L13 4 HEK AND REOVIRUS

=> D L13 IBIB ABS 1-4

L13 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2002:876030 CAPLUS
DOCUMENT NUMBER: 138:133772
TITLE: **Reovirus**-induced apoptosis requires mitochondrial release of Smac/DIABLO and involves reduction of cellular inhibitor of apoptosis protein levels
AUTHOR(S): Kominsky, Douglas J.; Bickel, Ryan J.; Tyler, Kenneth L.
CORPORATE SOURCE: Departments of Neurology and Medicine, University of Colorado Health Science Center, Denver, CO, 80262, USA
SOURCE: Journal of Virology (2002), 76(22), 11414-11424
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Many viruses belonging to diverse viral families with differing structure and replication strategies induce apoptosis both in cultured cells in vitro and in tissues in vivo. Despite this fact, little is known about the specific cellular apoptotic pathways induced during viral infection. We have previously shown that **reovirus**-induced apoptosis of **HEK** cells is initiated by death receptor activation but requires augmentation by mitochondrial apoptotic pathways for its maximum expression. We now show that **reovirus** infection of **HEK** cells is associated with selective cytosolic release of the mitochondrial proapoptotic factors cytochrome c and Smac/DIABLO, but not the release of apoptosis-inducing factor. Release of these factors is not associated with loss of mitochondrial transmembrane potential and is blocked by overexpression of Bcl-2. Stable expression of caspase-9b, a dominant-neg. form of caspase-9, blocks **reovirus**-induced caspase-9 activation but fails to significantly reduce activation of the key effector caspase, caspase-3. Smac/DIABLO enhances apoptosis through its action on cellular inhibitor of apoptosis proteins (IAPs). **Reovirus** infection is associated with selective down-regulation of cellular IAPs, including c-IAP1, XIAP, and survivin, effects that are blocked by Bcl-2 expression, establishing the dependence of IAP down-regulation on mitochondrial events. Taken together, these results are consistent with a model in which Smac/DIABLO-mediated inhibition of IAPs, rather than cytochrome c-mediated activation of caspase-9, is the key event responsible for mitochondrial augmentation of **reovirus**-induced apoptosis. These studies provide the 1st evidence for the association of Smac/DIABLO with virus-induced apoptosis.
REFERENCE COUNT: 63 THERE ARE 63 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2002:123164 CAPLUS
DOCUMENT NUMBER: 136:147504
TITLE: Method of producing infectious **reovirus**
INVENTOR(S): Coffey, Matthew C.; Thompson, Bradley G.
PATENT ASSIGNEE(S): Oncolytics Biotech, Inc., Can.
SOURCE: PCT Int. Appl., 30 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002012435	A1	20020214	WO 2001-CA1054	20010720
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2415749	AA	20020214	CA 2001-2415749	20010720
EP 1309672	A1	20030514	EP 2001-953084	20010720
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
BR 2001013122	A	20030722	BR 2001-13122	20010720
ZA 2003000410	A	20040126	ZA 2003-410	20010720
JP 2004505623	T2	20040226	JP 2002-517726	20010720
NZ 523510	A	20040827	NZ 2001-523510	20010720
US 2002037576	A1	20020328	US 2001-920012	20010802
US 6528305	B2	20030304		
US 2003166253	A1	20030904	US 2003-337911	20030108
US 6703232	B2	20040309		
US 2004126869	A1	20040701	US 2003-734552	20031211
PRIORITY APPLN. INFO.:			US 2000-224026P	P 20000810
			WO 2001-CA1054	W 20010720
			US 2001-920012	A1 20010802
			US 2003-337911	A3 20030108

AB A simple and efficient method of producing mammalian **reovirus** is developed using **HEK** 293 cells. The method provides for fast production of **reovirus** in high yield. Furthermore, this method provides for a simpler purification procedure of the produced **reovirus**.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2002:88971 CAPLUS
DOCUMENT NUMBER: 136:324098
TITLE: Advanced granulation technology (AGT): An alternate format for serum-free, chemically-defined and protein-free cell culture media
AUTHOR(S): Fike, Richard; Dadey, Barbara; Hassett, Richard; Radominski, Robert; Jayme, David; Cady, David
CORPORATE SOURCE: Cell Culture Research and Development, GIBCO/Invitrogen Corporation, Grand Island, NY, 14072, USA
SOURCE: Cytotechnology (2001), 36(1-3), 33-39
CODEN: CYTOER; ISSN: 0920-9069
PUBLISHER: Kluwer Academic Publishers
DOCUMENT TYPE: Journal
LANGUAGE: English

AB To overcome limitations of conventional milling technol., we investigated the application of fluid bed granulation for the production of dry-form nutrient media. Serum-free, protein-free and chemical-defined specialty media were produced in granulated format and compared with identical formulations manufactured by conventional methods. HPLC anal. of multiple lots of granulated materials demonstrated that biochem. constituents were precisely and homogeneously distributed throughout the granules and that nutrient levels were comparable to conventional formats. Comparison of medium performance in cell proliferation and biol. production assays demonstrated equivalence with reference media. The fluid bed granulation

process meets pharmaceutical quality requirements and may be applied to a broad range of nutrient formulations required for bioprodn. applications.

REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1974:401905 CAPLUS
DOCUMENT NUMBER: 81:1905
TITLE: Initiation of DNA replication in mammalian cells and its inhibition by **reovirus** infection
AUTHOR(S): Hand, Roger; Tamm, Igor
CORPORATE SOURCE: Rockefeller Univ., New York, NY, USA
SOURCE: Journal of Molecular Biology (1974), 82(2), 175-83
CODEN: JMOBAK; ISSN: 0022-2836
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The autoradiog. determined intervals between initiation sites on mammalian DNA were irregular, the modal interval being 40-50 μm, and were increased by **reovirus** infection. The mean distances between initiation sites on the DNA of mouse L929, hamster BHK, bovine MDBK, monkey CV1, and human **HEK** cells were 45.4, 30.1, 17.3, 42.2, and 22.7 resp. Initiation events on adjacent DNA strands were partially synchronized.

Most Recent Queries
Time Result

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#51
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11:34:46
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#50
Search recombinant human reovirus
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#49
Search recombinant reovirus and human
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#48
Search recombinant reovirus and serotype
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#45
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#43

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11:24:54
2169

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#37
Search poggioli 2000
10:01:18
18

#36
Search pogiolli 2000

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=> reovirus
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=> reassorted and L1
L2           2 REASSORTED AND L1

=> recombinant (l) L1
L3         164 RECOMBINANT (L) L1

=> "human embryo kidnay 293"
L4           0 "HUMAN EMBRYO KIDNAY 293"

=> "HEK 293"
L5        8825 "HEK 293"

=> L3 and L5
L6           0 L3 AND L5

=> L5 and L3
L7           0 L5 AND L3

=> L1 and L5
L8           4 L1 AND L5

=> D IBIB ABS 1-4
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